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1 **Neuromuscular fatigue and recovery after heavy resistance, jump, and sprint**
2 **training**

3

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10

11 **Short title:**

12 Neuromuscular fatigue after training

13

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25

27 **Purpose.** Training methods that require maximal intensity efforts against light- and
28 heavy-resistance are commonly used for athletic development. Typically these
29 sessions are separated by at least 48 hours recovery on the assumption that such
30 efforts elicit marked fatigue of the central nervous system (CNS), but this posit has
31 not been well-studied. The aim of the study was to assess the aetiology and recovery
32 of fatigue after heavy-resistance (strength), jump, and sprint training methods.

33 **Methods.** Ten male athletes completed three training sessions requiring maximal
34 efforts that varied in their loading characteristics; i) heavy resistance exercise (10×5
35 back squats at 80% 1RM) (STR); ii) jumping exercise (10×5 jump squats) (JUMP);
36 iii) maximal sprinting (15×30 m) (SPR). Pre-, post- and at 24, 48 and 72 h post-
37 participants completed a battery of tests to measure neuromuscular function using
38 electrical stimulation of the femoral nerve, and single- and paired-pulse magnetic
39 stimulation of the motor cortex, with evoked responses recorded from the knee
40 extensors. Fatigue was self-reported at each time point using a visual analogue scale.

41 **Results.** Each intervention elicited fatigue that resolved by 48 (JUMP) and 72 h (STR
42 & SPR). Decrements in muscle function (reductions in the potentiated quadriceps
43 twitch force) persisted for 48 h after all exercise. Reductions in voluntary activation
44 were present for 24 h after JUMP and SPRINT, and 48 h after STR. No other
45 differences in CNS function were observed as a consequence of training. **Conclusion.**
46 Strength, jump, and sprint training requiring repeated maximum efforts elicits fatigue
47 that requires up to 72 h to fully resolve, but this fatigue is not primarily underpinned
48 by decrements in CNS function.

49 **Key words.** Neurophysiology; brain; muscle; transcranial magnetic stimulation;
50 central nervous system

51

Introduction

52 Athletic development in a range of sports is characterized by the application of
53 various training means and methods in order to target specific adaptations. Resistance
54 training is a key training means employed by coaches and athletes to improve the
55 strength, impulse and speed qualities necessary for success in sports requiring
56 movements underpinned by high force and/or velocity. The methods by which
57 resistance training can be employed in an athlete's training programme can vary
58 depending on the desired adaptive outcome. For example, to target maximum
59 strength, coaches will typically utilize heavier loads (80-95% of 1 repetition
60 maximum (RM)) with consequent slower velocities of movement (1). Conversely, to
61 target the ability to produce high levels of force rapidly, submaximal loads are
62 required in order to accrue impulse quickly (2). To train acceleration and maximum
63 velocity running characteristics, the most effective training means is practice of
64 sprinting itself (3). Each of these training stimuli impose distinct demands on the
65 athlete, but their specific consequences are not well-studied or understood.

66

67 Heavy resistance and high velocity training methods typically require athletes to
68 repeatedly produce maximal efforts in order to stimulate adaptation. An inevitable
69 consequence of this is fatigue, a symptom or percept characterised by sensations of
70 tiredness and weakness (4). Fatigue is a complex phenomenon, and while likely
71 underpinned by a range of physiological and psychological mediators, an often-cited
72 posit amongst athletic development professionals is that repeated maximal efforts
73 elicit a high degree of “neuromuscular” or “central” fatigue, requiring prolonged (>48
74 hours) recovery. Such a postulate has also recently been cited in the academic
75 literature (5), further propagating this idea, despite a lack of peer-reviewed evidence.

76 Neuromuscular fatigue could feasibly relate to any alteration in the physiological
77 processes governing central nervous system (CNS) or muscle function, but is
78 typically quantified by examining voluntary and artificially-evoked forces during an
79 isometric muscle action. Peripheral neuromuscular fatigue refers to impairments in
80 muscle distal to the neuromuscular junction, quantified as a reduction in the resting
81 involuntary twitch response to nervous tissue stimulation (6). Central neuromuscular
82 fatigue is attributable to the central nervous system inadequately being able to activate
83 muscle to the required level, quantified as a reduction in voluntary activation (6).
84 Adjustments in CNS function can also be quantified via studying the evoked
85 responses to motor cortical stimulation (7). Single- and paired-pulse magnetic
86 stimulation of the motor cortex has been previously applied to understand acute and
87 chronic adjustments in CNS function in response to strength training (8-12) and
88 fatiguing single-limb (13-15) and locomotor exercise (16). In concert, the application
89 of these techniques to study adjustments in neuromuscular function after athletic
90 training could help explain the etiology of fatigue, and aid practitioners in the
91 appropriate scheduling of, and recovery from, different training methods.

92

93 While decrements in neuromuscular function, particularly of the CNS, are widely
94 considered when programming training stimuli, the evidence underpinning the idea
95 that heavy strength and power sessions require >48 h recovery is incomplete. Previous
96 studies recently demonstrated that heavy resistance exercise elicited greater acute
97 reductions in voluntary force than a similar low-resistance, high-velocity “power”
98 session (17), and that these heavy resistance exercise induced decrements persisted at
99 24 h post-exercise in elite athletes (18). Bartolomei *et al.* (19) recently demonstrated
100 greater and more prolonged strength and jump performance impairments after

101 “hypertrophy” style training (higher volume, lower load, shorter rest periods)
102 compared to a training stimulus targeting strength development (lower volume, higher
103 intensity, longer rest periods). Collectively these findings suggest the acute and
104 prolonged adjustments underpinning the fatigue experienced after resistance exercise
105 varies between training methods, but these studies were limited by both the range of
106 outcome measures studied, and/or a limited profile of the time-course recovery of
107 neuromuscular function. Further study is warranted to comprehensively assess the
108 acute and prolonged neuromuscular adjustments induced by the typical training
109 means and methods commonly employed in the physical preparation of athletes. Such
110 information will be of high value to practitioners when prescribing training stimuli.

111

112 The aim of the study was to assess the etiology and recovery of neuromuscular fatigue
113 in response to heavy resistance, jumping, and sprinting exercise. It was hypothesised
114 that the maximal nature of all exercise interventions would induce marked
115 neuromuscular fatigue that would require >48 hours to resolve, and that the time-
116 course of recovery would be similar between interventions.

117

118 **Methods**

119 **Participants**

120 Ten male participants (age 21 ± 2 years, stature, 1.82 ± 0.05 m, mass, 85 ± 12 kg)
121 gave their written, informed consent to participate in the study, which was approved
122 by the Northumbria University Faculty of Health & Life Sciences Ethics Committee.
123 All participants had >3 years history of training experience utilising resistance and
124 maximal speed methods, and were currently competing in intermittent ($n = 6$), or
125 track and field ($n = 4$) sports at University or national standard.

126

127 **Design**

128 Participants initially visited the laboratory on two separate occasions for preliminary
129 assessments and to habituate to the measurement tools of the study. Subsequent to
130 this participants completed three experimental trials, each spanning four consecutive
131 days and separated by one week, in a randomised, counterbalanced order. On the first
132 day of each experimental trial, participants completed one of three interventions as
133 follows: i) a heavy resistance exercise session consisting of repeated sets of back
134 squats (STR); ii) a low-load, high-velocity exercise session consisting of repeated sets
135 of jump squats (JUMP); iii) a maximal speed training session consisting of repeated
136 30 m sprints (SPR). Pre-, immediately post-, and at 24, 48 and 72 h post- a battery of
137 assessments to measure fatigue and neuromuscular function were administered. Prior
138 to all visits participants were instructed to refrain from caffeine (24 hours), alcohol
139 (48 hours), and to arrive 2 h post-prandial in a fully rested, hydrated state. Participants
140 were also instructed not to perform any exercise other than that required by the study
141 for the duration of their participation. To account for any potential detraining-induced
142 changes in physical fitness, a “refresh” session consisting of maintenance loads for
143 the physical qualities under study was employed between experimental trials. An
144 overview of the experimental trials can be viewed in Supplemental Digital Content 1.

145

146 **Procedures**

147 *Practice trial*

148 Prior to the experimental trials, participants visited the laboratory on two occasions
149 for habituation to the measurement tools of the study (on both visits), and an
150 assessment of 1 repetition maximum (1RM) back squat strength or jump squat

151 performance (on separate visits). Prior to all exercise (practice & experimental trials)
152 participants completed a structured ten-minute warm-up, which incorporated jogging,
153 dynamic flexibility movements, mobility exercises specific to squatting, jumping, and
154 sprinting, and 3 × 30 m progressive strides at 70, 80 and 90% of perceived maximum
155 sprint speed. For the assessment of maximum isoinertial strength, participants first
156 completed warm-up sets of 3-5 repetitions of back squats (high bar position),
157 beginning with an unloaded barbell and progressing to 50%, 70%, 80% and 90% of
158 their estimated 1RM. The load on the bar was then incremented by 2-5% until
159 participants could not complete 1 repetition. The technical execution of each lift
160 required participants to descend under control (2 s tempo) to a depth where the femur
161 was parallel to the floor. Participants then immediately reversed the movement and
162 were instructed to maximally accelerate the bar during the concentric phase. A
163 repetition was deemed unsuccessful if participants could not complete the concentric
164 phase in ≤ 2 s. Maximum isoinertial strength was 126 ± 14 kg, or $150 \pm 15\%$ body
165 mass. For jump squats, participants completed vertical jumps for maximum height,
166 beginning with body mass (plus a wooden dowel) and incrementing by 5 kg; the first
167 increment was achieved by replacing the dowel with a lightweight training barbell
168 with a mass of 5 kg. Each repetition required participants to squat to a self-selected
169 depth (approximating a half squat) and jump for maximum height. Jump height was
170 recorded using photoelectronic timing gates (Optojump Next, Microgate, Milan, Italy)
171 for 2 to 3 efforts at each load. When participants were unable to maintain performance
172 within 5% of their unloaded jump height because of added resistance, the test was
173 terminated and the highest applied load where squat jump height was maintained was
174 used for experimental trials (mean, SD 10 ± 5 kg, with a range of 0 to 20 kg,
175 additional load).

176

177 *Experimental trials; exercise intervention*

178 On the first day of each experimental trial, subsequent to pre-test assessment,
179 participants completed one of three exercise prescriptions; i) heavy resistance training
180 consisting of 10×5 repetitions of the high bar back squat at 80% 1RM, with 3 min
181 recovery (STR); ii) 10×5 repetitions of jump squats, with 3 min recovery (JUMP);
182 iii) 15×30 m maximum sprints, with 2 min recovery (SPR). For STR and JUMP
183 participants were encouraged to maximally accelerate the load, and the velocity of
184 each repetition was monitored using a wearable linear position transducer (PushBand,
185 Heap Analytics, Toronto, Canada). For SPR participants began each sprint 0.5 m
186 behind the first timing gate, and were encouraged to sprint maximally through the
187 timing gate at 30 m. Each sprint was measured using photocell technology (TC
188 Timing system, Brower Timing Systems, Draper, Utah, USA). For all trials
189 participants were provided feedback on the execution of each repetition to promote a
190 maximum effort. Post-training, participants were asked for a whole trial session rating
191 of perceived exertion (RPE) using the 0-10 category ratio scale. While it was
192 impossible to equate training load between the experimental trials, the configurations
193 for STR, JUMP and SPR were designed in consultation with experienced strength and
194 conditioning coaches to represent a “heavy” stimulus for the physical quality under
195 stress, and were similar in duration (approximately 45 min, including the standardised
196 warm-up).

197

198 *Experimental trials; outcome measures*

199 On each occasion participants completed a battery of assessments to measure fatigue
200 and neuromuscular function. All outcome measures were assessed pre-, post-, and at
201 24, 48 and 72 h post-exercise, unless otherwise stated.

202

203 *Visual analogue scales & creatine kinase*

204 Upon arrival, and post-exercise after assessment of neuromuscular function,
205 participants completed visual analogue scales (VAS, 100 mm scale) to record fatigue
206 and perceptions of muscle soreness. For fatigue the VAS was anchored with the
207 verbal descriptors “not fatigued at all” to “extremely fatigued”; participants were
208 asked to rate their general feeling of “fatigue, tiredness, weakness and lethargy”. For
209 muscle soreness the VAS was anchored with “no soreness” to “extremely sore”;
210 participants preceded their rating with three repetitions of a body weight squat and
211 were asked to rate their “muscle soreness and pain”. Subsequent to this fingertip
212 samples of capillary blood were obtained and immediately assayed for creatine kinase
213 (CK) concentration (Reflotron, Roche Diagnostics, Germany).

214

215 *Assessment of neuromuscular function*

216 The evoked force and electromyographic (EMG) responses of the *rectus femoris* (RF)
217 to transcranial magnetic stimulation (TMS) of the primary motor cortex, and electrical
218 stimulation of the femoral nerve, were used to assess neuromuscular fatigue,
219 corticospinal excitability, and the status of inhibitory intracortical networks. The
220 assessment of neuromuscular function took place subsequent to perceptual
221 assessments and capillary blood sampling at all time points except for post-exercise,
222 where it was conducted first in order to capture the extent of neuromuscular fatigue
223 elicited by the exercise intervention.

224

225 A calibrated load cell (MuscleLab force sensor 300, Ergotest technology, Norway)
226 recorded muscle force (N) during an isometric maximal voluntary contraction
227 (iMVC) of the knee extensors. During contractions, participants sat with hips and
228 knees at 90° flexion, with a load cell fixed to a custom-built chair and attached to the
229 participants right leg, superior to the ankle malleoli, with a noncompliant cuff.
230 Electrical activity from the RF and *biceps femoris* (BF) were recorded from surface
231 electrodes (Ag/AgCl; Kendall H87PG/F, Covidien, Mansfield, MA, USA) placed 2
232 cm apart over the belly of each muscle, with a reference electrode placed on the
233 patella. Electrode placement was marked with indelible ink to ensure consistent
234 placement throughout the study, with the areas cleaned and shaved prior to electrode
235 placement. The electrodes recorded the root-mean-square (RMS) amplitude for sub-
236 maximal and maximal voluntary contractions, the compound muscle action potential
237 (M-wave) from the electrical stimulation of the femoral nerve, and the motor evoked
238 potential (MEP) elicited by TMS. Signals were amplified: gain $\times 1000$ for EMG and
239 $\times 300$ for force (CED 1902; Cambridge Electronic Design, Cambridge, UK), band-
240 pass filtered (EMG only: 20-2000 Hz), digitized (4 kHz; CED 1401, Cambridge
241 Electronic Design) and analysed offline. Further details on these methods are
242 provided below.

243

244 *Motor nerve stimulation*

245 Motor nerve stimulation was used for the measurement of contractile function, muscle
246 membrane excitability and voluntary activation (VA). Single electrical stimuli were
247 administered using square wave pulses (200 μ s) via a constant-current stimulator
248 (DS7AH, Digitimer Ltd., Hertfordshire, UK) using self-adhesive surface electrodes

249 (Nidd Valley Medical Ltd., North Yorkshire, UK). Electrical stimuli were first
250 administered to the motor nerve at rest in 20 mA step-wise increments from 20 mA
251 until the maximum quadriceps twitch amplitude (Q_{tw} , N) and muscle compound
252 action potential (M_{max} , mV) were elicited. To ensure a consistent, supramaximal
253 stimulus and account for any activity-induced changes in axonal excitability, the
254 resulting stimulation intensity was increased by 30% for all subsequent stimulus. The
255 peak-to-peak amplitude and area of the electrically evoked maximal compound action
256 potential (M_{max}) was used as a measure of membrane excitability. Participants
257 subsequently completed six iMVCs (3-5 s duration) of the knee extensors, separated
258 by 60 s rest. For the final three iMVCs, electrical stimuli were delivered during and 2
259 s post contraction to assess VA and potentiated quadriceps twitch force ($Q_{tw,pot}$)
260 respectively.

261

262 *Motor cortical stimulation*

263 Single- and paired-pulse TMS of 1 ms duration were delivered using a concave
264 double cone coil using two linked monopulse magnetic stimulators (Magstim 200,
265 The Magstim Company Ltd, Whitland, UK). The junction of the double cone coil was
266 aligned tangentially to the sagittal plane, with its centre 1-2 cm to the left of the
267 vertex. The optimal coil placement was determined at the start of each trial as the
268 position that elicited the largest MEP in the RF, with a concomitant small MEP in the
269 BF. The position was marked with indelible ink for consistent placement during
270 subsequent trials. The stimulator intensity was based on active motor threshold
271 (AMT) measured during a 10% iMVC. In order to determine AMT, the stimulator
272 intensity was increased in 5% steps beginning at 35% of stimulator output until a
273 consistent MEP with peak-to-peak amplitudes of $>200 \mu V$ was found. Thereafter,

274 stimulus intensity was reduced in 1% step until an MEP of >200 μ V was found in
275 50% of stimulations.

276

277 *Corticospinal excitability & Short-interval intracortical inhibition (SICI)*

278 Once AMT was established, the stimulator intensities required to assess the MEP
279 response to varying TMS intensities (stimulus-response curve) were determined in
280 order to assess corticospinal excitability. Participants held a submaximal voluntary
281 contraction (10% iMVC) with one set of five stimuli delivered at each of 90%, 100%,
282 110%, 120%, 130%, 140%, 150% and 160% of AMT in a randomized and
283 counterbalanced order, with 4-6 s between each stimuli and 15 s between each set.
284 For SICI, ten single and ten paired-pulse TMS stimuli were administered in two sets
285 of 10 stimuli during a 10% iMVC, for measurement of unconditioned and conditioned
286 MEP amplitude respectively. Paired-pulse TMS consisted of a subthreshold
287 conditioning pulse at 70% of AMT, and a suprathreshold test pulse at 120% AMT,
288 with an inter-stimulus interval (ISI) of 2 ms. Single- and paired-pulses (\times 10 each)
289 were delivered in a pre-determined randomised order, with 4-6 s between each
290 stimulation and a short rest between each set. This assessment was conducted pre-
291 exercise, and at 24 hour intervals thereafter until 72 h post.

292

293 *Voluntary activation with TMS*

294 Single pulse TMS was delivered during brief (3-5 s) contractions at 100%, 75% and
295 50% iMVC, separated by 5 s of rest, for determination of voluntary activation with
296 TMS (VA_{TMS}). This procedure was repeated 3 times with 15 s rest between each set.
297 The stimulation intensity was set at the stimulator output that elicited the maximum
298 superimposed twitch force (SIT) during a 50% iMVC. The SIT force elicited from

299 contractions at 100%, 75%, and 50% were used to determine VA_{TMS} (see data
300 analysis section for details).

301

302 *Experimental trials: “refresh session”*

303 On the final day of each experimental trial, after all outcome measures had been
304 completed, a “refresh” session designed to maintain the physical qualities under study
305 over the course of the experimental period was employed. This consisted of a low-
306 volume, high-intensity stimulus for each physical quality in a single session (3×5
307 sets of back squats at 80% 1RM, 3×5 maximal effort jump squats, 3×30 m maximal
308 effort sprints). Previous research has demonstrated that strength qualities can be
309 adequately maintained for prolonged periods using low doses provided the intensity
310 of exercise remains close to maximal (20, 21).

311

312 **Data analysis**

313 Voluntary activation assessed through the interpolated twitch technique (22) was
314 quantified by comparing the amplitude of the superimposed twitch force to the
315 potentiated twitch (100 Hz) delivered 2 s following the iMVC at rest using the
316 following equation: Motor point VA (%) = $[1 - (SIT/Q_{tw, pot}) \times 100]$. Voluntary
317 activation using TMS (VA_{TMS}) was assessed during contractions at 50%, 75% and
318 100% iMVC using linear regression of the superimposed twitch force evoked by TMS
319 (23), with the regression analysis confirming a linear relationship at each time-point
320 (r^2 range = 0.89 ± 0.03 to 0.95 ± 0.04). The estimated resting twitch (ERT) was
321 calculated as the y-intercept of the linear regression between the mean amplitude of
322 the SIT force evoked by TMS at each contraction intensity. Subsequently, VA_{TMS} was
323 quantified using the equation $[1 - (SIT/ERT) \times 100]$. To quantify SICI, the ratio of

324 the average conditioned paired-pulse MEP was expressed relative to the average
325 unconditioned MEP at 120% AMT. Recruitment curves were constructed by plotting
326 the TMS stimulation intensity relative to AMT against the MEP amplitude averaged
327 from the five stimulations at each intensity, expressed relative to M_{\max} . The ratio of
328 the MEP amplitude to the maximum M-wave was used as an index of corticospinal
329 excitability. In order to provide a summary measure of corticospinal excitability, the
330 summated area under the stimulus-response curve was calculated for each participant
331 at each time point using the trapezoid integration method (24). The root mean square
332 EMG amplitude (RMS_{EMG}) and average force was calculated in the 80 ms prior to
333 each TMS to ensure a similar level of background muscle activity was present during
334 the stimulus-response curve and SICI measurements. The peak-to-peak amplitude of
335 evoked MEP and M_{\max} were measured offline.

336

337 **Statistical analysis**

338 Data are presented as mean \pm SD. To ascertain the time-course recovery of
339 neuromuscular fatigue within-trial, one-way repeated measures ANOVA across time
340 were employed for STR, JUMP and SPR data. Significant main effects were followed
341 up with Dunnett's multiple comparison procedure, with the pre-exercise score used as
342 the control category. To assess between-trial differences in the magnitude of
343 neuromuscular fatigue induced by STR, JUMP and SPR, two-way (trial \times time)
344 factorial repeated measures ANOVA analysis was employed. As baseline scores did
345 not differ between trials for any outcome measure, significant trial \times time interaction
346 effects were followed up with one-way repeated measures ANOVA, and *post-hoc*
347 Tukey-adjusted pairwise comparisons at each time point to locate statistically
348 significant between-trial differences. The assumptions underpinning these statistical

349 procedures were verified as per the guidelines outlined by Newell *et al.* (25). Data
350 were analysed using GraphPad Prism (version 7, GraphPad Software Inc., La Jolla,
351 CA). Statistical significance was accepted at $P < 0.05$.

352

353

354

Results

355 **Exercise responses.** All participants successfully completed the prescribed training
356 interventions. For STR, the load lifted was 101 ± 11 kg. Repetition velocity decreased
357 from $0.53 \text{ m}\cdot\text{s}^{-1}$ in set 1, to $0.44 \text{ m}\cdot\text{s}^{-1}$ in set 10 ($P < 0.05$), with a best of 0.54 ± 0.07
358 $\text{m}\cdot\text{s}^{-1}$ and worst of $0.41 \pm 0.07 \text{ m}\cdot\text{s}^{-1}$ independent of set. Session RPE averaged 8 ± 2
359 for STR. For JUMP, mean repetition velocity was successfully maintained throughout
360 the exercise ($1.61 \pm 0.17 \text{ m}\cdot\text{s}^{-1}$ in set 1 vs. $1.56 \pm 0.14 \text{ m}\cdot\text{s}^{-1}$ in set ten, $P = 0.31$, best
361 score of $1.69 \pm 0.11 \text{ m}\cdot\text{s}^{-1}$, worst of $1.48 \pm 0.10 \text{ m}\cdot\text{s}^{-1}$) and session RPE was lower (5
362 ± 1) than STR ($P = 0.001$). For SPR, 40 m sprint time declined from 4.40 ± 0.14 s in
363 set 1 to 4.55 ± 0.22 s in set fifteen ($P = 0.04$), with a fastest sprint of 4.36 ± 0.16 s and
364 a slowest of 4.61 ± 0.24 s. Session RPE after SPR (6 ± 2) was not different to STR (P
365 $= 0.18$) or JUMP ($P = 0.33$)

366

367 **Perceived fatigue & muscle damage responses.** All exercise interventions elicited
368 significant perceived fatigue (Table 1) that persisted for 48 h after STR (48 h, $P =$
369 0.002) and SPR training (48 h, $P = 0.008$), and 24 h after JUMP training (24 h, $P =$
370 0.02). Between trials, both STR and SPR training resulted in greater perceived fatigue
371 than JUMP training for up to 48 h (Figure 1, panel A). Similar patterns were also
372 evident for perceptions of muscle soreness; all training resulted in increases in muscle
373 soreness that were different to baseline for 48 h, and between trials - both STR (for up

374 to 72 h, $P = 0.0006$) and SPR (for up to 48 h, $P = 0.0008$) elicited a greater magnitude
375 of soreness in comparison to JUMP (Figure 1, panel B). Creatine kinase peaked at 24
376 h in all trials and was different to baseline for 24, 48 and 72 h for STR, JUMP and
377 SPR respectively (Table 1). Between trials, CK was lower at 24 h in JUMP compared
378 to both STR ($P = 0.001$) and SPR ($P = 0.002$) (Figure 1, panel C).

379

380 **Neuromuscular fatigue.** All exercise interventions resulted in declines in iMVC
381 force that took until 72 h to fully resolve in all trials (Table 2). The magnitude of the
382 reduction in iMVC force immediately post-exercise was higher after STR compared
383 to JUMP ($P < 0.001$) and SPR ($P < 0.001$), a difference that persisted at 24 hours (P
384 = 0.02 and 0.05 respectively, Figure 2, panel A). Reductions in VA were also evident
385 immediately post-exercise for all trials, and persisted for 48 h after STR ($P = 0.004$),
386 and 24 h after JUMP ($P = 0.015$) and SPR ($P = 0.023$, Table 2). Significant reductions
387 in VA_{TMS} were also evident post-exercise in all trials (all $P < 0.05$), but returned to
388 baseline quicker than VA; by 48 h in STR and 24 h in JUMP and SPR (Table 2). The
389 magnitude of reductions in VA, measured with both motor nerve and motor cortical
390 stimulation, was not different between exercise interventions (Figure 2, panel B & C).
391 All trials resulted in reductions in $Q_{tw,pot}$, that took 72 h to fully resolve (Table 2).
392 Between trials there were larger reductions in $Q_{tw,pot}$ immediately-post STR compared
393 to both JUMP and SPR (both $P < 0.001$), with no differences between trials thereafter
394 (Figure 2, panel D).

395

396 **Corticospinal excitability and SICI.** Exercise resulted in no modulation of
397 corticospinal excitability (Figure 3, stimulus-response curves) or SICI (Figure 4), both
398 within and between trials (all $P > 0.05$). The EMG_{RMS} was also not different within

399 and between trials (supplementary material, Table 3). For a full list of surface EMG
400 responses to TMS and electrical stimulation please see supplementary material, Table
401 3.

402

403

404

Discussion

405 The aim of the study was to assess the effect of strength, jump and sprint training,
406 performed with maximal intent, on the etiology and time-course of neuromuscular
407 fatigue and recovery. In accordance with our hypothesis, all training stimuli resulted
408 in neuromuscular adjustments that took up to 72 h to fully resolve. For twitch force,
409 indicative of peripheral fatigue, strength training resulted in larger post-exercise
410 reductions compared to jump and sprint training, but the time-course recovery was
411 similar thereafter, with marked decrements still evident at 48 h post-exercise in all
412 trials. Reductions in voluntary activation, an indicator of central fatigue, persisted for
413 24 h after jump and sprint training, and 48 h after strength training, with no difference
414 between trials in the magnitude of these reductions. Measures of CNS responsiveness
415 and inhibition were not modulated in response to the training stimuli at any time
416 point. Perceptual indicators of fatigue and soreness followed a similar time-course of
417 recovery to measures of neuromuscular function, requiring up to 72 h to return to
418 baseline, with a tendency for jump training to be less fatiguing compared to strength
419 and sprint training. Collectively these data indicate that maximal intent, relatively
420 high volume, strength, jump and sprint training methods elicit neuromuscular fatigue,
421 mediated by both central and peripheral mechanisms, that requires up to 72 h to fully
422 resolve.

423

424 **Time-course of recovery of neuromuscular fatigue after training.** An often-cited
425 posit in strength and conditioning is the idea that training methods performed with
426 maximal intent, such as those studied here, result in central fatigue, or are CNS
427 intensive, and require 48-72 h recovery before similarly intense stimuli are imposed
428 (26, 5, 27). To date however, the formal study of neuromuscular fatigue in the days
429 post-training has been limited (19, 17, 18, 28, 29). Here we show that strength, jump
430 and sprint training elicits marked neuromuscular central and peripheral fatigue, that
431 can require up to 72 h to fully resolve, which provides some support to these previous
432 assertions. The capacity to produce voluntary force was impaired for 48 h after all
433 training, with decrements in MVC force of 8%, 7% and 6% on average for strength,
434 jump and sprint training. Similarly, twitch force was reduced compared to baseline for
435 48 h in all trials, indicating a prolonged decrement in muscle function, with values
436 remaining depressed by 5-6% on average at 48 h. Reductions in voluntary activation
437 persisted for 48 h after strength training, and 24 h after jump and sprint training,
438 suggesting heavy resistance training elicited more prolonged central fatigue than the
439 other methods studied. At the 48 h time point the decrement in voluntary activation
440 averaged 5%, 2% and 3% for strength, jump and sprint training respectively.
441 Collectively, these data suggest that neuromuscular fatigue after training methods that
442 emphasise maximal intent is persistent, and multi-factorial. This underscores the need
443 for appropriate recovery between such sessions, alongside interventions that address
444 the multi-factorial nature of fatigue. The data also provide some support to the
445 assertion that training sessions that emphasise maximal intent should be separated by
446 at least 48 h if peak performance is a priority, as the majority of variables under study
447 took 72 h to fully resolve.

448

449 **“Central” fatigue after training.** Fatigue of the CNS is often implicated as a
450 primary consideration after training modes that emphasise maximal intent, and recent
451 reviews have called for an increased emphasis on the recovery of central and “brain”
452 fatigue after exercise (30, 31). However, the formal study, and precise definition, of
453 what constitutes central fatigue is limited. Here we specifically measured central
454 fatigue as a reduction in the ability of the CNS to activate skeletal muscle. This
455 activation deficit was evident post-training for up to 24 h after jump and sprint
456 training, and up to 48 h after heavy resistance training. We also measured variables
457 purported to reflect CNS excitability and inhibition, but these did not modulate with
458 training. In contrast, the capacity to produce voluntary force was impaired for 48 h in
459 all trials, decrements in muscle function (indicative of peripheral fatigue) persisted for
460 48 h in all trials, and sensory perceptions of fatigue and soreness persisted for 48-72 h
461 post. The magnitude of central fatigue was also modest, with voluntary activation
462 returning to within 5% of baseline in the majority of cases (n = 6, 8 & 6 respectively
463 for strength, jump and sprint training) by 24 h post. Additionally, the magnitude of the
464 decrement post-trial was similar to that previously observed in our lab for prolonged
465 cycling exercise (32, 33), repeated-sprint exercise (34) and simulated intermittent-
466 sprint exercise (35). The recovery of central neuromuscular fatigue in the days post-
467 was also similar to that observed after simulated intermittent-sprint exercise (35).
468 Therefore, the idea that recovery of the CNS should be prioritised after methods of
469 training that emphasise maximal intent is debatable, but perhaps simply reflects an
470 imprecise definition of terms. Fatigue is a symptom, or percept, characterised by
471 sensations of tiredness and weakness (4), underpinned by a myriad of physiological
472 and psychological mechanisms; what is commonly perceived as central fatigue by
473 athletes and coaches is likely more accurately interpreted as fatigue *per se*. That is,

474 the feelings of tiredness and weakness that athletes experience in the days post-
475 exercise are likely underpinned by a range of mechanisms relating to both central and
476 peripheral function, and not primarily attributable to “CNS” fatigue. A caveat to this
477 conclusion is the acknowledgement that our ability to measure aspects of CNS
478 function, and thus infer the impact of exercise, is limited by the available
479 measurement tools. For example, even the most widely acknowledged measure of
480 central fatigue - a reduction in voluntary activation of skeletal muscle – has
481 questionable validity (36). This notwithstanding, our data suggest that the fatigue
482 experienced after the training methods under study is multi-factorial and not primarily
483 underpinned by central mechanisms.

484

485 **Differential effect of strength, jump and sprint training.** A number of differences
486 were observed between trials that indicated the jumping training stimulus elicited less
487 fatigue, and took less time to recover from. These included differential effects on
488 iMVC and twitch force, the creatine kinase response, and perceptions of fatigue and
489 muscle soreness, in comparison to heavy resistance exercise and sprinting. However,
490 whether these differences could be primarily attributed to differences in the force-
491 velocity requirements of the differing sessions is debatable. Both the heavy resistance
492 (back squat to parallel depth) and sprinting stimuli required greater displacement of
493 load (external or body mass) in comparison to power training (jumping from a half
494 squat). The ostensibly increased work required during STR and SPR (and associated
495 metabolic demand), and the increased potential for muscle damage at longer muscle
496 lengths, could explain the differences observed between trials independent of
497 differences in the force-velocity demands of the exercise. Equating the training
498 stimulus between trials is an impossible endeavour, and therefore any between-trial

499 comparisons should be interpreted with caution. However, the relatively lower stress
500 and quicker recovery observed after jumping compared to heavy resistance training is
501 not without precedent. Howatson *et al.* (18) previously observed strength training
502 (consisting of 4 × 5 heavy back squat, split squat and push press) elicited reductions
503 in iMVC for up to 24 h, whereas the same session conducted with lower loads and
504 higher repetition velocities elicited no reduction in iMVC. Additionally, Linnamo *et*
505 *al.* (29) previously demonstrated a higher degree of acute neuromuscular fatigue
506 following heavy load vs. light load “explosive” bilateral leg extension resistance
507 training. These previous data, and the current study, indicate that training methods
508 that emphasise the ability to generate impulse to accelerate relatively light loads
509 might require less recovery time than heavy resistance or maximal sprint training, a
510 finding that has implications for the scheduling of such activities.

511

512 **Corticospinal excitability and short intracortical inhibition.** There were no
513 discernible adjustments in corticospinal excitability nor short intracortical inhibition
514 at any time point in response to all exercise interventions. Corticospinal excitability
515 has been shown to modulate acutely with single limb fatiguing exercise (13-15) and
516 ballistic isometric exercise (9), and chronically after single limb (8, 12) and whole
517 body (10) resistance training programmes. Short intracortical inhibition has similarly
518 been demonstrated to be modulated after a period of resistance training (10), and
519 acutely during locomotor exercise (16) and after fatiguing isometric knee extensor
520 exercise (37). Of importance, these acute adjustments seem to quickly resolve upon
521 exercise cessation (37, 16); this could explain why, in the present study, we did not
522 observe any differences post-exercise as the measurement of these variables was
523 delayed in comparison to previous work. The finding that neither corticospinal

524 excitability nor short intracortical inhibition were modulated with recovery in the days
525 post-exercise concurs with previous studies from our laboratory studying the etiology
526 and recovery of neuromuscular fatigue after simulated and competitive intermittent-
527 sprint exercise (38, 35). Thus, while measures of CNS excitability and inhibition
528 might be modulated during and immediately post-exercise, or chronically in response
529 to longer-term training, they do not systematically differ from baseline in the days
530 post-fatiguing exercise.

531

532 In addition to an inability to match training stimuli between trials, the ecological
533 validity of both the imposed sessions, and the measurement protocols, could also be
534 questioned. Considering the primary variables under study (i.e. indicators of
535 neuromuscular fatigue), we deliberately chose to study a high volume of exercise for
536 each training stimulus, and limited each to a single exercise that required a significant
537 contribution from the quadriceps muscle group, and where possible were
538 biomechanically similar (e.g. back squats vs. jump squats). For these reasons, the
539 applicability of the results to regular athletic development training, which typically
540 involves lower volumes and higher variation of exercises within sessions, is
541 questionable. There are of course unlimited configurations of exercise selection, sets,
542 repetitions and recovery durations that could be manipulated, and consequently any
543 decisions on the exercise intervention employed in a study of this nature could be
544 questioned. Additionally, adjustments in neuromuscular function as a consequence of
545 exercise were studied during single-limb, isometric knee extensor muscle actions.
546 This assessment set-up is required to measure neuromuscular fatigue, however these
547 adjustments might not fully reflect decrements in the type of dynamic knee extensor
548 function required of the exercise modes under study, and athletic performance more

549 generally. These limitations notwithstanding, the data do provide new information on
550 the nature of fatigue and recovery after resistance and speed training; an area of
551 research that is under-studied, and in need of further investigation.

552

553 In conclusion, this study has demonstrated that training methods requiring repeated
554 maximal intensity efforts elicit marked neuromuscular fatigue that requires up to 72 h
555 to fully resolve. The observed neuromuscular fatigue was of both a central and
556 peripheral origin, with a faster recovery of central, compared to peripheral,
557 neuromuscular fatigue. The data provide partial support for the idea that training
558 methods that emphasise maximal intent to express force or velocity should be
559 separated by at least 48 h, but the recovery of central nervous system function is not
560 necessarily the primary aim of this period. Rather, the residual fatigue experienced by
561 athletes after such training is multi-factorial, and thus development of appropriate
562 monitoring and rest/recovery strategies that reflect this is warranted. Further research
563 is required to further probe the consequences of maximal intensity training using
564 novel measurement tools, and stimuli that more accurately reflect the day-to-day
565 practice of different athletic groups.

566

567

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572

573

Conflict of Interest

574 The authors have no conflict of interest to declare. The results of the study do not
575 constitute endorsement by ACSM. The results of the study are presented clearly,
576 honestly, and without fabrication, falsification, or inappropriate data manipulation

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- 580 1. Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P.
581 Increased rate of force development and neural drive of human skeletal muscle
582 following resistance training. *J Appl Physiol (1985)*. 2002;93(4):1318-1326.
583
- 584 2. Cronin JB, McNair PJ, Marshall RN. Force-velocity analysis of strength-
585 training techniques and load: implications for training strategy and research. *J*
586 *Strength Cond Res*. 2003;17(1):148-155.
587
- 588 3. Rumpf MC, Lockie RG, Cronin JB, Jalilvand F. Effect of Different Sprint
589 Training Methods on Sprint Performance Over Various Distances: A Brief
590 Review. *J Strength Cond Res*. 2016;30(6):1767-1785.
591
- 592 4. Enoka RM, Duchateau J. Translating Fatigue to Human Performance. *Med Sci*
593 *Sports Exerc*. 2016;48(11):2228-2238.
594
- 595 5. Hartmann H, Wirth K, Keiner M, Mickel C, Sander A, Szilvas E. Short-term
596 Periodization Models: Effects on Strength and Speed-strength Performance.
597 *Sports Med*. 2015;45(10):1373-1386.
598
- 599 6. Carroll TJ, Taylor JL, Gandevia SC. Recovery of central and peripheral
600 neuromuscular fatigue after exercise. *J Appl Physiol (1985)*.
601 2017;122(5):1068-1076.
602
- 603 7. Goodall S, Howatson G, Romer L, Ross E. Transcranial magnetic stimulation
604 in sport science: A commentary. *Eur J Sport Sci*. 2014;14(sup1):S332-S340.
605
- 606 8. Griffin L, Cafarelli E. Transcranial magnetic stimulation during resistance
607 training of the tibialis anterior muscle. *J Electromyogr Kinesiol*.
608 2007;17(4):446-452.
609
- 610 9. Nuzzo JL, Barry BK, Gandevia SC, Taylor JL. Acute Strength Training
611 Increases Responses to Stimulation of Corticospinal Axons. *Med Sci Sports*
612 *Exerc*. 2016;48(1):139-150.
613
- 614 10. Weier AT, Pearce AJ, Kidgell DJ. Strength training reduces intracortical
615 inhibition. *Acta Physiol (Oxf)*. 2012;206(2):109-119.
616
- 617 11. Zult T, Goodall S, Thomas K, Hortobágyi T, Howatson G. Mirror illusion
618 reduces motor cortical inhibition in the ipsilateral primary motor cortex during
619 forceful unilateral muscle contractions. *J Neurophysiol*. 2015;113(7):2262-
620 2270.
621
- 622 12. Zult T, Goodall S, Thomas K, Solnik S, Hortobágyi T, Howatson G. Mirror
623 Training Augments the Cross-education of Strength and Affects Inhibitory
624 Paths. *Med Sci Sports Exerc*. 2016;48(6):1001-1013.
625

- 626 13. Goodall S, Howatson G, Thomas K. Modulation of specific inhibitory
627 networks in fatigued locomotor muscles of healthy males. *Exp Brain Res.*
628 2018;236(2):463-473.
629
- 630 14. Takahashi K, Maruyama A, Hirakoba K et al. Fatiguing intermittent lower
631 limb exercise influences corticospinal and corticocortical excitability in the
632 nonexercised upper limb. *Brain Stimul.* 2011;4(2):90-96.
633
- 634 15. Williams PS, Hoffman RL, Clark BC. Cortical and spinal mechanisms of task
635 failure of sustained submaximal fatiguing contractions. *PLoS One.*
636 2014;9(3):e93284.
637
- 638 16. Sidhu SK, Lauber B, Cresswell AG, Carroll TJ. Sustained cycling exercise
639 increases intracortical inhibition. *Med Sci Sports Exerc.* 2013;45(4):654-662.
640
- 641 17. Brandon R, Howatson G, Strachan F, Hunter AM. Neuromuscular response
642 differences to power vs strength back squat exercise in elite athletes. *Scand J*
643 *Med Sci Sports.* 2015;25(5):630-639.
644
- 645 18. Howatson G, Brandon R, Hunter AM. The Response to and Recovery From
646 Maximum-Strength and -Power Training in Elite Track and Field Athletes. *Int*
647 *J Sports Physiol Perform.* 2016;11(3):356-362.
648
- 649 19. Bartolomei S, Sadres E, Church DD et al. Comparison of the recovery
650 response from high-intensity and high-volume resistance exercise in trained
651 men. *Eur J Appl Physiol.* 2017;117(7):1287-1298.
652
- 653 20. Bickel CS, Cross JM, Bamman MM. Exercise dosing to retain resistance
654 training adaptations in young and older adults. *Med Sci Sports Exerc.*
655 2011;43(7):1177-1187.
656
- 657 21. Ronnestad BR, Nymark BS, Raastad T. Effects of in-season strength
658 maintenance training frequency in professional soccer players. *J Strength*
659 *Cond Res.* 2011;25(10):2653-2660.
660
- 661 22. Merton PA. Voluntary strength and fatigue. *J Physiol.* 1954;123(3):553-564.
662
- 663 23. Todd G, Taylor JL, Gandevia SC. Measurement of voluntary activation of
664 fresh and fatigued human muscles using transcranial magnetic stimulation. *J*
665 *Physiol.* 2003;551(Pt 2):661-671.
666
- 667 24. Carson RG, Nelson BD, Buick AR, Carroll TJ, Kennedy NC, Cann RM.
668 Characterizing changes in the excitability of corticospinal projections to
669 proximal muscles of the upper limb. *Brain Stimul.* 2013;6(5):760-768.
670
- 671 25. Newell J, Aitchison T, Grant S. *Statistics for Sports and Exercise Science: A*
672 *practical approach.* Harlow: Pearson Education; 2010. 427
673
- 674 26. Gambetta V. *Athletic development: The art & science of functional sports*
675 *conditioning.* New World Library; 2007. p. 71

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692
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705
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717
718
719
720
721
722
723

27. Olbrecht J. *The science of winning: planning, periodizing and optimizing swim training*. F&G Partners; 2000. p. 5-15
28. Johnston M, Cook CJ, Crewther BT, Drake D, Kilduff LP. Neuromuscular, physiological and endocrine responses to a maximal speed training session in elite games players. *Eur J Sport Sci*. 2015;15(6):550-556.
29. Linnamo V, Hakkinen K, Komi PV. Neuromuscular fatigue and recovery in maximal compared to explosive strength loading. *Eur J Appl Physiol Occup Physiol*. 1998;77(1-2):176-181.
30. Minett GM, Duffield R. Is recovery driven by central or peripheral factors? A role for the brain in recovery following intermittent-sprint exercise. *Front Physiol*. 2014;5:24.
31. Rattray B, Argus C, Martin K, Northey J, Driller M. Is it time to turn our attention toward central mechanisms for post-exertional recovery strategies and performance? *Front Physiol*. 2015;6:79.
32. Thomas K, Elmeua M, Howatson G, Goodall S. Intensity-Dependent Contribution of Neuromuscular Fatigue after Constant-Load Cycling. *Med Sci Sports Exerc*. 2016;48(9):1751-1760.
33. Thomas K, Goodall S, Stone M, Howatson G, St Clair Gibson A, Ansley L. Central and peripheral fatigue in male cyclists after 4-, 20-, and 40-km time trials. *Med Sci Sports Exerc*. 2015;47(3):537-546.
34. Goodall S, Charlton K, Howatson G, Thomas K. Neuromuscular fatigability during repeated-sprint exercise in male athletes. *Med Sci Sports Exerc*. 2015;47(3):528-536.
35. Thomas K, Dent J, Howatson G, Goodall S. Etiology and Recovery of Neuromuscular Fatigue after Simulated Soccer Match Play. *Med Sci Sports Exerc*. 2017;49(5):955-964.
36. Contessa P, Puleo A, De Luca CJ. Is the notion of central fatigue based on a solid foundation? *J Neurophysiol*. 2016;115(2):967-977.
37. Hunter SK, McNeil CJ, Butler JE, Gandevia SC, Taylor JL. Short-interval cortical inhibition and intracortical facilitation during submaximal voluntary contractions changes with fatigue. *Exp Brain Res*. 2016;234(9):2541-2551.
38. Brownstein CG, Dent JP, Parker P et al. Etiology and Recovery of Neuromuscular Fatigue following Competitive Soccer Match-Play. *Front Physiol*. 2017;8:831.

724

Tables & Figures

725 **Table 1.** Within-trial differences in fatigue and perceptions of muscle soreness
726 measured using visual analogue scales (100 mm scale), and creatine kinase (CK),
727 measured pre- and in the 72 h post-strength, jump, and sprint training. Values are
728 mean \pm SD. * = significant difference within-trial from pre-test score.

729

730 **Table 2.** Within-trial differences in isometric maximum voluntary contraction
731 strength and measures of neuromuscular fatigue pre-, post, and 24, 48, and 72 hours
732 post-strength, jump and sprint training. Values are mean \pm SD. * = significant
733 difference from pre-test score within trial.

734

735 **Figure 1.** Between-trial differences in fatigue (A), muscle soreness (B) and creatine
736 kinase (C) measured pre-, post- and 24, 48 and 72 hours post- strength, jump, and
737 sprint training. Between trial differences indicated by * = difference between strength
738 and jump; # = difference between jump and sprint; ^ = difference between strength
739 and sprint (all $P > 0.05$). Individual responses are plotted, with lines representing the
740 mean score.

741

742 **Figure 2.** Between-trial differences in isometric maximum voluntary contraction
743 force (A), voluntary activation measured with motor nerve (B) and motor cortical (C)
744 stimulation, and quadriceps potentiated twitch force (D) Between trial differences
745 indicated by * = difference between strength and jump; # = difference between jump
746 and sprint; ^ = difference between strength and sprint (all $P > 0.05$). Individual
747 responses are plotted, with lines representing the mean score.

748

749 **Figure 3.** Motor evoked potential (expressed relative to Maximum M-wave) stimulus-
750 response curves measured above and below active motor threshold (AMT, 100%) pre-
751 , and 24, 48 and 72 hours post- strength (A), jump (B) and sprint (C) training. Values
752 are mean \pm SD. A reference line is included at 60% to assist comparison between
753 trials.

754

755 **Figure 4.** Short intracortical inhibition (SICI) expressed as the ratio between
756 conditioned and unconditioned motor evoked potentials pre-, and 24, 48 and 72 hours
757 post- strength, jump and sprint training. Individual responses are plotted, with lines
758 representing the mean score.

759

760

Supplemental digital content

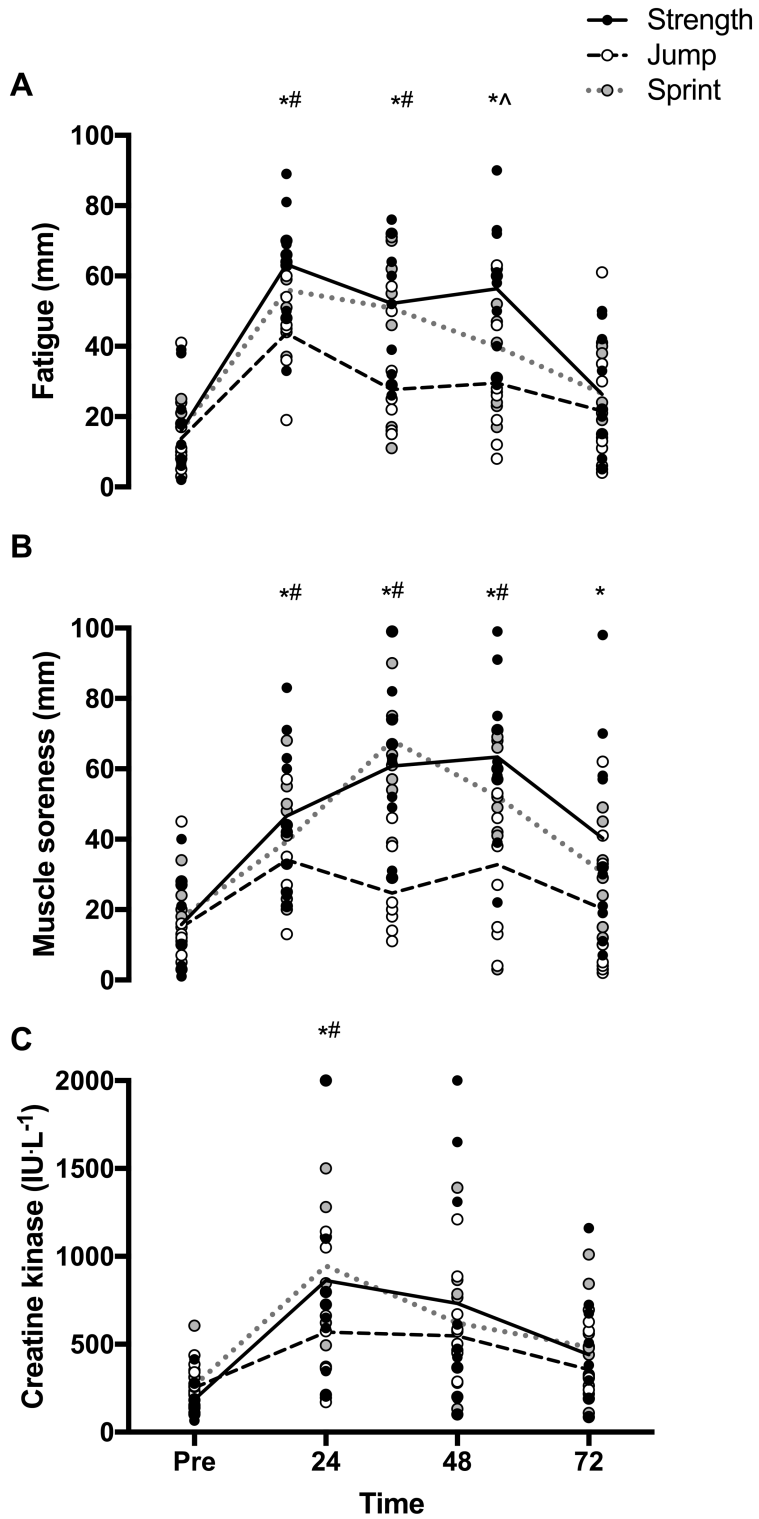
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762 **Supplemental digital content 1.pdf.** Schematic of experimental protocol. Pre-
763 exercise and at 24, 48 and 72 h post participants completed the battery of assessments
764 in the same order. After the pre-exercise assessment participants completed one of
765 three exercise interventions: i) heavy resistance training consisting of 10×5
766 repetitions of the high bar back squat at 80% 1RM, with 3 min recovery (STR); ii) 10
767 $\times 5$ repetitions of a jump squat, with 3 min recovery (JUMP); iii) 15×30 m
768 maximum sprints, with 2 min recovery (SPR). Participants were encouraged to
769 complete every repetition with maximal intensity. Immediately post-exercise, central
770 and peripheral neuromuscular fatigue were evaluated within 2 min of exercise
771 cessation. Pre-exercise and at 24 h intervals thereafter, single-pulse transcranial
772 magnetic stimulation (TMS) were administered during a submaximal isometric
773 contraction at various percentages (90 to 160%) of active motor threshold (AMT) for

774 the assessment of corticospinal excitability. Paired-pulse TMS were administered
775 during submaximal contraction for assessment of short intracortical inhibition.

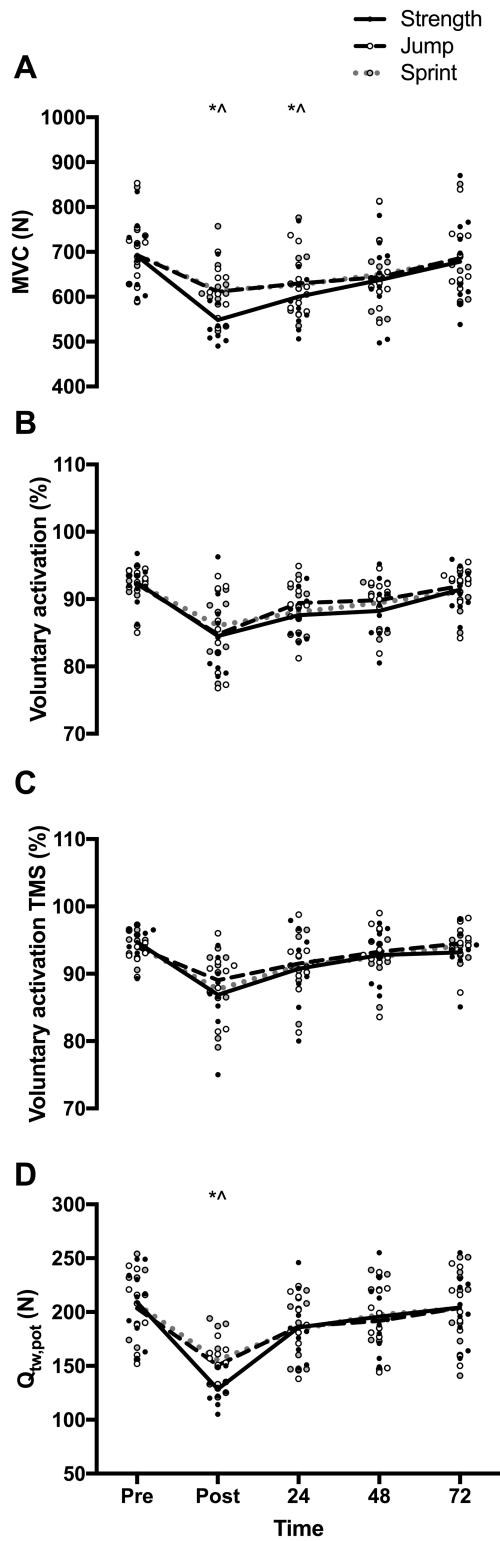
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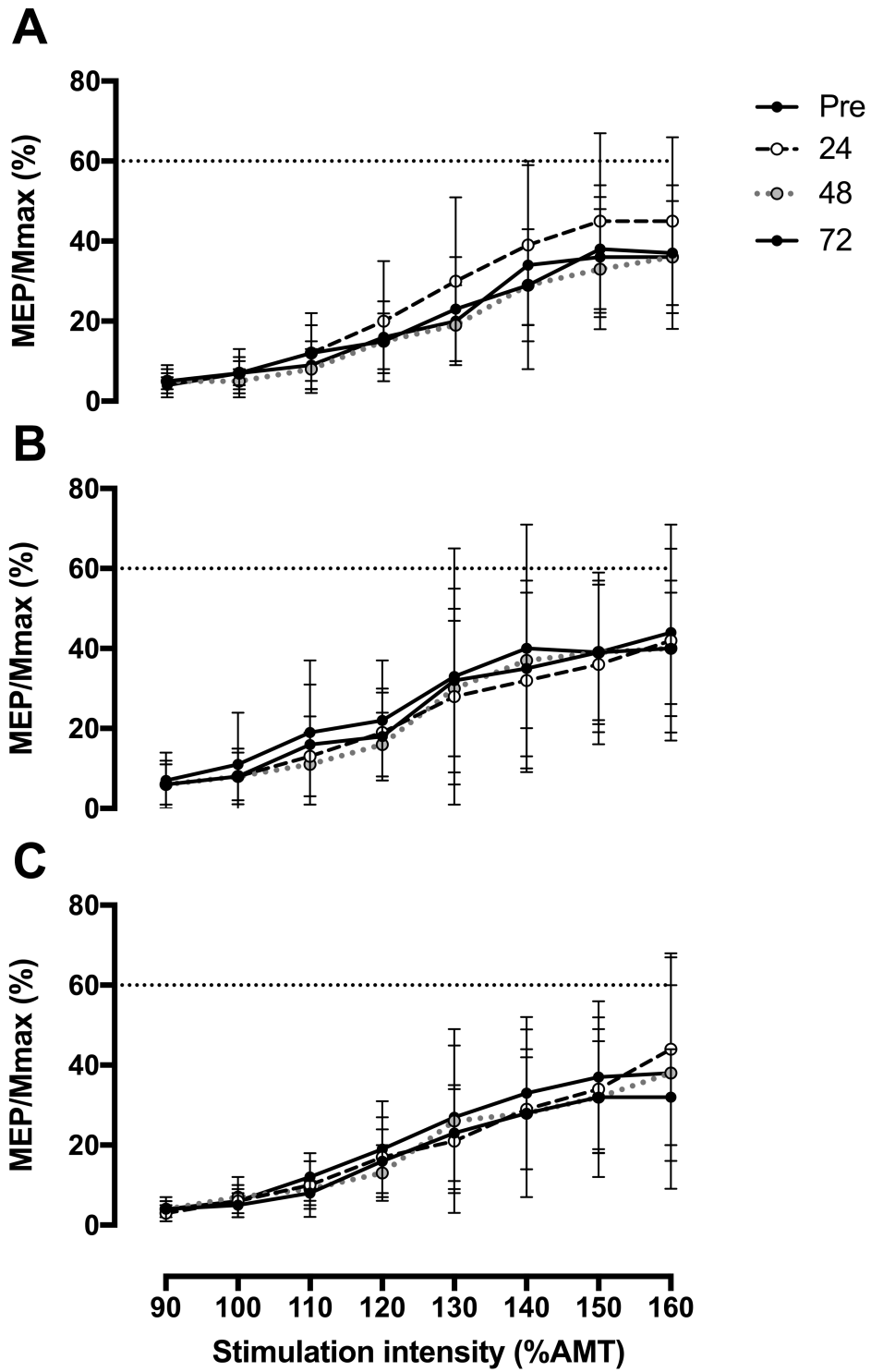
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779 Figure 1.



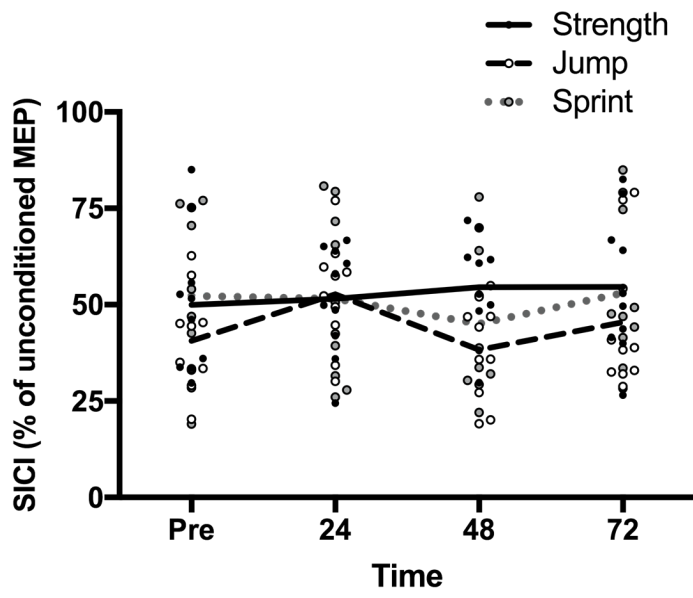
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781 Figure 2



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783 Figure 3



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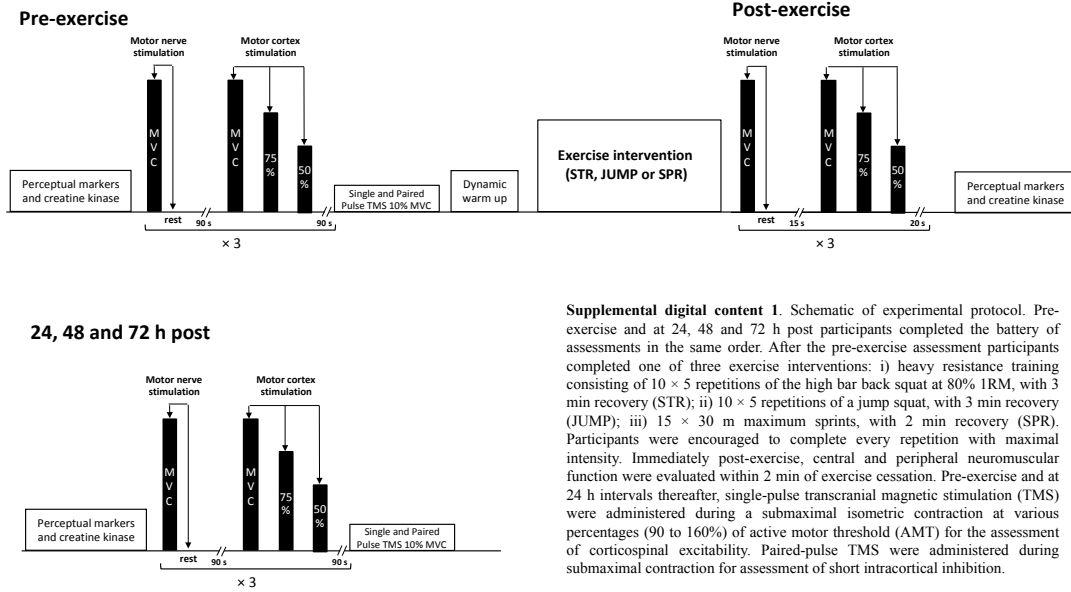
785 Figure 4

Table 1. Within-trial differences in fatigue and perceptions of muscle soreness measured using visual analogue scales (100 mm scale), and creatine kinase (CK), measured pre- and in the 72 h post-strength, jump, and sprint training. Values are mean \pm SD. * = significant difference within-trial from pre-test score.

	Strength	Jump	Sprint
Fatigue (mm)			
Pre-	16 \pm 13	14 \pm 11	16 \pm 6
Post-	63 \pm 16*	44 \pm 15*	56 \pm 11*
24 h	52 \pm 19*	28 \pm 15*	51 \pm 21*
48 h	56 \pm 19*	30 \pm 16	40 \pm 16*
72 h	26 \pm 16	22 \pm 17	27 \pm 13
Muscle soreness (mm)			
Pre-	16 \pm 13	15 \pm 13	18 \pm 9
Post-	47 \pm 22*	34 \pm 10*	39 \pm 17*
24 h	61 \pm 22*	25 \pm 11*	68 \pm 17*
48 h	63 \pm 23*	33 \pm 21*	52 \pm 21*
72 h	40 \pm 29	20 \pm 21	31 \pm 18
CK (IU·L⁻¹)			
Pre-	185 \pm 98	253 \pm 114	265 \pm 142
24 h	863 \pm 659*	569 \pm 340*	946 \pm 531*
48 h	733 \pm 673	547 \pm 328*	622 \pm 357*
72 h	440 \pm 333	356 \pm 205	484 \pm 270*

Table 2. Within-trial differences in isometric maximum voluntary contraction strength and measures of neuromuscular fatigue pre-, post, and 24, 48, and 72 hours post-strength, jump and sprint training. Values are mean \pm SD. * = significant difference from pre-test score within trial.

	Strength	Jump	Sprint
iMVC (N)			
Pre-	691 \pm 78	693 \pm 78	693 \pm 74
Post-	548 \pm 61*	611 \pm 52*	614 \pm 66*
24	600 \pm 78*	630 \pm 63*	627 \pm 72*
48	637 \pm 90*	644 \pm 77*	650 \pm 83*
72	678 \pm 102	686 \pm 77	682 \pm 78
VA (%)			
Pre-	92.4 \pm 2.9	92.2 \pm 2.7	92.3 \pm 2.6
Post-	84.5 \pm 5.8*	84.8 \pm 6.1*	86.1 \pm 4.7*
24	87.6 \pm 3.3*	89.4 \pm 3.8*	88.1 \pm 3.5*
48	88.2 \pm 4.4*	89.9 \pm 3.8	89.5 \pm 3.3
72	91.4 \pm 3.2	92.0 \pm 3.2	91.1 \pm 2.9
VA_{TMS} (%)			
Pre-	94.7 \pm 2.5	94.0 \pm 2.4	94.2 \pm 2.0
Post-	86.9 \pm 5.7*	89.1 \pm 4.7*	87.7 \pm 5.3*
24	90.7 \pm 5.7*	91.5 \pm 5.1	91.2 \pm 4.0*
48	92.8 \pm 4.1	93.3 \pm 4.4	92.5 \pm 3.4
72	93.2 \pm 3.5	94.5 \pm 3.3	94.2 \pm 2.0



Supplemental digital content 1. Schematic of experimental protocol. Pre-exercise and at 24, 48 and 72 h post participants completed the battery of assessments in the same order. After the pre-exercise assessment participants completed one of three exercise interventions: i) heavy resistance training consisting of 10×5 repetitions of the high bar back squat at 80% 1RM, with 3 min recovery (STR); ii) 10×5 repetitions of a jump squat, with 3 min recovery (JUMP); iii) 15×30 m maximum sprints, with 2 min recovery (SPR). Participants were encouraged to complete every repetition with maximal intensity. Immediately post-exercise, central and peripheral neuromuscular function were evaluated within 2 min of exercise cessation. Pre-exercise and at 24 h intervals thereafter, single-pulse transcranial magnetic stimulation (TMS) were administered during a submaximal isometric contraction at various percentages (90 to 160%) of active motor threshold (AMT) for the assessment of corticospinal excitability. Paired-pulse TMS were administered during submaximal contraction for assessment of short intracortical inhibition.